

RESEARCH MONOGRAPH . COMPOUND PROFILE

Coenzyme Q10 (Ubiquinol)

An endogenous redox cofactor of the mitochondrial electron transport chain, declining with age and statin use, sold as a dietary supplement.

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COMPOUND CLASS	Endogenous mitochondrial cofactor

ABSTRACT

Coenzyme Q10 (CoQ10, ubiquinone; CAS 303-98-0; molecular formula $C_{59}H_{90}O_4$; molecular weight 863.34) is an endogenous redox cofactor essential for mitochondrial electron transport between Complex I/II and Complex III. The compound is synthesized from tyrosine and the mevalonate pathway; statin use reduces endogenous synthesis through HMG-CoA reductase inhibition (the same pathway used for cholesterol synthesis). Plasma and tissue CoQ10 decline with age. Supplementation has been studied for cardiovascular function (heart failure, hypertension), statin-associated muscle symptoms, and migraine prophylaxis. Bioavailability of the oxidized form (ubiquinone) is poor; the reduced form (ubiquinol) has 3- to 5-fold better bioavailability and is the preferred supplemental form for elderly subjects with reduced redox capacity. Doses are typically 100 to 400 mg per day of ubiquinol or higher of ubiquinone.

1. Compound Identification

Coenzyme Q10 (Ubiquinol) is described as endogenous mitochondrial cofactor. An endogenous redox cofactor of the mitochondrial electron transport chain, declining with age and statin use, sold as a dietary supplement.

1.1 Class summary

Endogenous mitochondrial cofactor

1.2 Subtitle

An endogenous redox cofactor of the mitochondrial electron transport chain, declining with age and statin use, sold as a dietary supplement.

2. Abstract

Coenzyme Q10 (CoQ10, ubiquinone; CAS 303-98-0; molecular formula C₅₉H₉₀O₄; molecular weight 863.34) is an endogenous redox cofactor essential for mitochondrial electron transport between Complex I/II and Complex III. The compound is synthesized from tyrosine and the mevalonate pathway; statin use reduces endogenous synthesis through HMG-CoA reductase inhibition (the same pathway used for cholesterol synthesis). Plasma and tissue CoQ10 decline with age. Supplementation has been studied for cardiovascular function (heart failure, hypertension), statin-associated muscle symptoms, and migraine prophylaxis. Bioavailability of the oxidized form (ubiquinone) is poor; the reduced form (ubiquinol) has 3- to 5-fold better bioavailability and is the preferred supplemental form for elderly subjects with reduced redox capacity. Doses are typically 100 to 400 mg per day of ubiquinol or higher of ubiquinone.

3. Mechanism of Action

Endogenous electron transport chain cofactor; statin-suppressed synthesis. Antioxidant in lipid environments.

The mechanistic profile described above represents the consensus position from peer-reviewed literature where available; for compounds with sparse formal characterization, this section reflects vendor-grade reports and structurally-related class pharmacology and should be interpreted with appropriate caution.

4. Pharmacokinetics and Routes of Administration

Pharmacokinetic data are summarized within the abstract for compounds with formal Phase 1 records. Compounds without published human pharmacokinetic studies are noted as such; investigators should treat the absence of data as a research limitation.

5. Reported Research Dose Ranges

100 to 400 mg per day of ubiquinol; higher of ubiquinone.

Doses are scaled from clinical use where the compound is approved as a medicine, from Phase 2/3 trials where development advanced, or from rodent dose-response data scaled by allometric assumptions where the compound has no human clinical record. Investigators should treat dose ranges from research-chemical-grade vendors as starting points for their own pharmacokinetic and tolerability characterization rather than validated dosing recommendations.

6. Sourcing and Quality Verification

Investigators should obtain analytical confirmation of identity (mass spectrometry; for peptides, additionally amino acid analysis or N-terminal sequencing) and purity (HPLC area-percent, ideally greater than 95 percent) on every lot. Vendor certificates of analysis vary in quality; third-party analytical confirmation through independent laboratories (e.g., Janoshik Analytical, Anabolic Lab) is the gold standard for research-grade peptides and small molecules.

For compounds with established pharmaceutical-grade marketing in any jurisdiction, sourcing from the marketed product (where legal and accessible) provides the highest quality assurance.

7. Reconstitution and Handling

Peptides should be reconstituted in bacteriostatic water or sterile water for injection in the volume appropriate to the desired concentration; lyophilized peptides should be allowed to come to room temperature before reconstitution to minimize precipitation. Reconstituted peptides should be stored refrigerated and used within the timeframe specified by the manufacturer (typically 14 to 30 days for bacteriostatic water reconstitutions, shorter for sterile water).

Small molecules: storage conditions vary by compound; consult the certificate of analysis for the specific product. Most are stable at room temperature in dry, dark conditions; oxidation-sensitive compounds (e.g., catecholamine precursors) require cool storage and protection from light.

8. Stack Interaction Considerations

Investigators combining this compound with other research substances should consider receptor-level interactions (additive effects at shared targets), enzyme-level interactions (CYP-mediated drug-drug interactions where applicable), and pharmacodynamic interactions (additive sedation, additive serotonergic activity, additive hemodynamic effects).

The stack interaction section in published monographs of well-characterized compounds is necessarily incomplete for any specific combination; investigators should treat novel combinations with elevated caution and titrate gradually.

9. References

1. Mortensen SA, et al. The effect of coenzyme Q10 on morbidity and mortality in chronic heart failure (Q-SYMBIO). JACC Heart Fail 2014.

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